Endocardial wavefront propagation guiding atrioventricular nodal re-entrant tachycardia ablation after modified Fontan

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Introduction

Atrioventricular nodal re-entrant tachycardia (AVNRT) is the most common type of supraventricular tachycardia (SVT) in older children and adults.[1] Successful treatment can often be achieved by means of catheter ablation (ABL) targeting the right inferior extension of the atrioventricular node within the triangle of Koch (TOK), the so-called slow pathway (SP), thereby eliminating the substrate for re-entry. The longstanding "electroanatomic" approach involves delivering ABL applications at sites in the inferoposterior TOK, where typical SP electrograms (EGMs) are recorded. Sometimes additional ABL is performed in nearby sites or in a linear fashion from the tricuspid annulus back to and sometimes within the proximal coronary sinus (CS).[2]

Although this approach is generally successful when treating patients with structurally normal hearts, the advent of 3-dimensional (3D) electrophysiology (EP) mapping has led to alternatives to improve precision of SP localization before ABL, particularly important for children in whom fewer total ABL lesions may be desired, and for patients with unusual anatomy related to congenital heart disease (CHD).[3] One such approach involves 3D mapping within the TOK during sinus rhythm to uncover a low-voltage bridge that could reflect the SP.[4–6] More recently, dense 3D mapping in patients with structurally normal hearts has facilitated visualization of relevant wavefront propagation patterns,[7] and successful treatment can be achieved by ablating the site immediately superior to a zone of wavefront collision within the TOK.[7,8] Interestingly, this site can include a small patch of abrupt conduction slowing suspected to reflect conduction behavior as impulses penetrate the SP from higher in the atrial septum.[9]

KEY TEACHING POINTS

- Preoperative review of all imaging, including available 3-dimensional imaging is critical for planning safe and effective intracardiac access in patients with complex anatomy.
- Use of high-density electrophysiology mapping may be useful for ablating typical and atypical arrhythmia substrates in patients with complex congenital heart disease.
- Endocardial wavefront propagation and identification of a zone of abrupt conduction slowing may aid in the ablation of atrioventricular nodal re-entrant tachycardia, both in structurally normal hearts, but especially in patients with congenital heart disease.

Patients with postoperative univentricular hearts present special challenges to successful ABL for AVNRT, given the distorted anatomy, limited transcatheter access, atypical endocardial atrial EGMs, and ectopic atrial rhythms. We describe our experience with such a patient having recurrent and refractory AVNRT. The ABL procedure involved access via transhepatic and transbaffle punctures, and successful and uncomplicated ABL was guided by endocardial wavefront propagation.

Case report

A 27-year-old woman with complex CHD developed recurrent bouts of SVT causing syncope and requiring multiple emergency department visits. Her CHD included hypoplastic left heart syndrome with left atrial isomerism, bilateral superior vena cavae, including persistent left superior vena cava to CS, and interrupted inferior vena cava with azygous continuation. Computed tomography imaging confirmed bilateral left lung morphologies and polysplenia. She had undergone Norwood procedure, pulmonary artery stenting, and staged

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Kawashima Fontan palliation. Progressive cyanosis from multiple pulmonary arteriovenous malformations prompted surgical implantation of a conduit to connect central hepatic veins to the azygous vein, which later required transcatheter stenting (Figure 1).

Available electrocardiogram recordings of her SVT showed a narrow complex long RP tachycardia that was adenosine-sensitive in the emergency department (Figure 2A). Diagnostic EP study in 2018 using transesophageal and retrograde-transaortic catheters suggested the SVT mechanism was atypical AVNRT. Catheter access to the atrial chambers was unavailable at that time, so ABL was not pursued. Unfortunately, she continued to have frequent SVT recurrences despite beta-blockers and flecainide. After review of echocardiograms and advanced imaging indicated a transhepatic and transbaffle approach was feasible, she was referred for possible ABL procedure.

After induction of general anesthesia, our interventional team used a Versacross dilator/sheath system (Boston Scientific, Marlborough, MA) to achieve transhepatic access to the central hepatic vein confluence, then crossed the Fontan baffle to enter the pulmonary venous side of the Fontan circuit (Figure 1). After review of all prior imaging, including preoperative computed tomography scan of the heart, this approach was considered the most feasible route for intracardiac access and providing a relatively "straight shot" for manipulation of EP catheters. This long deflectable sheath was used to interchangeably deploy intracardiac mapping and ABL EP catheters; a transesophageal EP catheter was used for temporal reference during EP testing and mapping. A D/F EZ Steer mapping/ABL catheter (Biosense Webster, Diamond Bar, CA) was used to create 3D geometries of the pulmonary venous chamber, which included the medial right atrium, residual atrial septum, TOK, dilated CS os, and tricuspid annulus, and to pass an EP catheter into the right ventricle for intermittent ventricular pacing. With atrial pacing protocols at baseline, AV block cycle length was 320 milliseconds, there was no PR>RR phenomenon, antegrade AV conduction curves were smooth, and the atrioventricular nodal effective refractory period was <600/250 milliseconds. There was no change in the QRS morphology suggestive of twin AV nodes either during EP testing or on any prior ECG recordings. With ventricular pacing, ventriculoatrial (VA) block cycle length was 500 milliseconds. On isoproterenol (1 μg/min), VA block cycle length shortened to 360 milliseconds, VA decrement was seen with ventricular extrastimulus protocols, and the VA conduction system effective refractory period was <550/260 milliseconds. Ventricular extrastimuli with triples (500/300/300/300 milliseconds) on isoproterenol repeatedly induced the patient's clinical long RP tachycardia (cycle length 300 milliseconds) (Figure 2B). There was no VA "jump" prior to induction of the tachycardia. Ventricular overdrive pacing into tachycardia advanced local atrial EGMs after the transition zone, followed by a V-A-V response when pacing was withheld, and postpacing interval– tachycardia cycle length was 196 milliseconds, all consistent with atypical AVNRT.[10]

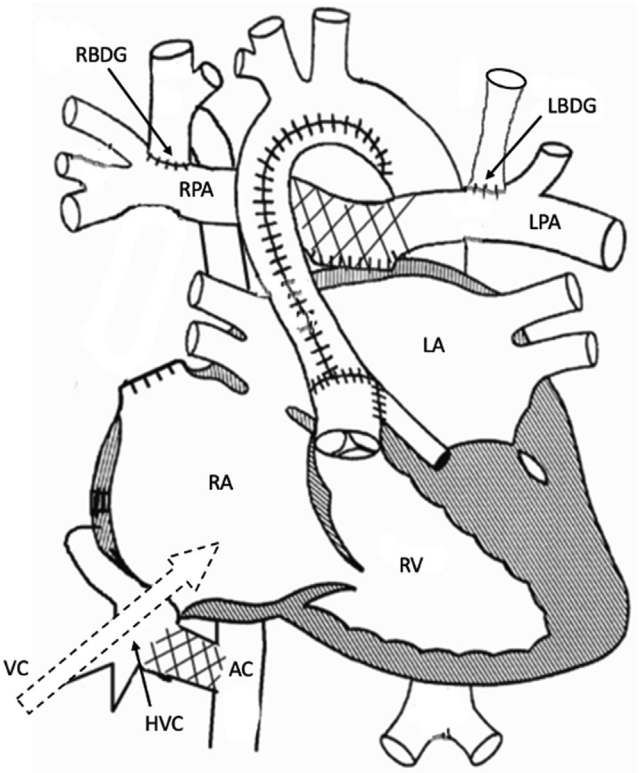

Figure 1 A 27-year-old patient with hypoplastic left heart syndrome, interrupted inferior vena cava with azygous continuation, and bilateral superior vena cavae status post multiple surgical palliations, including Norwood procedure, left pulmonary artery stenting, left-sided bidirectional Glenn anastomosis, and implantation of conduit connecting hepatic venous confluence to the azygous vein. Transhepatic and transbaffle puncture was required for intracardiac access to perform 3-dimensional mapping and catheter ablation of the slow pathway to the atrioventricular (AV) node to treat atypical AV node re-entry tachycardia. AC = azygous continuation; HVC = hepatic venous confluence; LA = left atrium; LBDG = left bidirectional Glenn; LPA = left pulmonary artery; RA = right atrium; RBDG = right bidirectional Glenn; RPA = right pulmonary artery; RV = right ventricle; VC = Versacross (Boston Scientific, Marlborough, MA) catheter access.

The EZ Steer catheter was exchanged for a multipolar OCTARAY mapping catheter (Biosense Webster) and dense endocardial 3D EP mapping was performed off isoproterenol during the patient's prevailing low right atrial rhythm to record atrial voltage and activation patterns and His bundle EGMs. Propagation mapping revealed a zone of wavefront collision in the inferior–mid TOK. Adjusting CARTO 3 (Biosense Webster) early-meets-late timing thresholds revealed a discrete patch of abrupt slowing immediately inferior to that (Figure 3, Supplemental Video 1), where local EGMs were consistent with possible SP potentials. Despite adjusting voltage display thresholds, a low-voltage "bridge" was not demonstrable in the TOK. Gentle radiofrequency (RF) application (20 W, 50°C) at the site of abrupt slowing below the collision zone induced rapid junctional rhythm, and RF was terminated immediately. There was no evidence for AV block or PR prolongation. Another RF application slightly lower in the TOK had a similar outcome. Interestingly, SVT was no longer inducible at baseline or on isoproterenol (up to 2 μg/min). The RF catheter was exchanged for

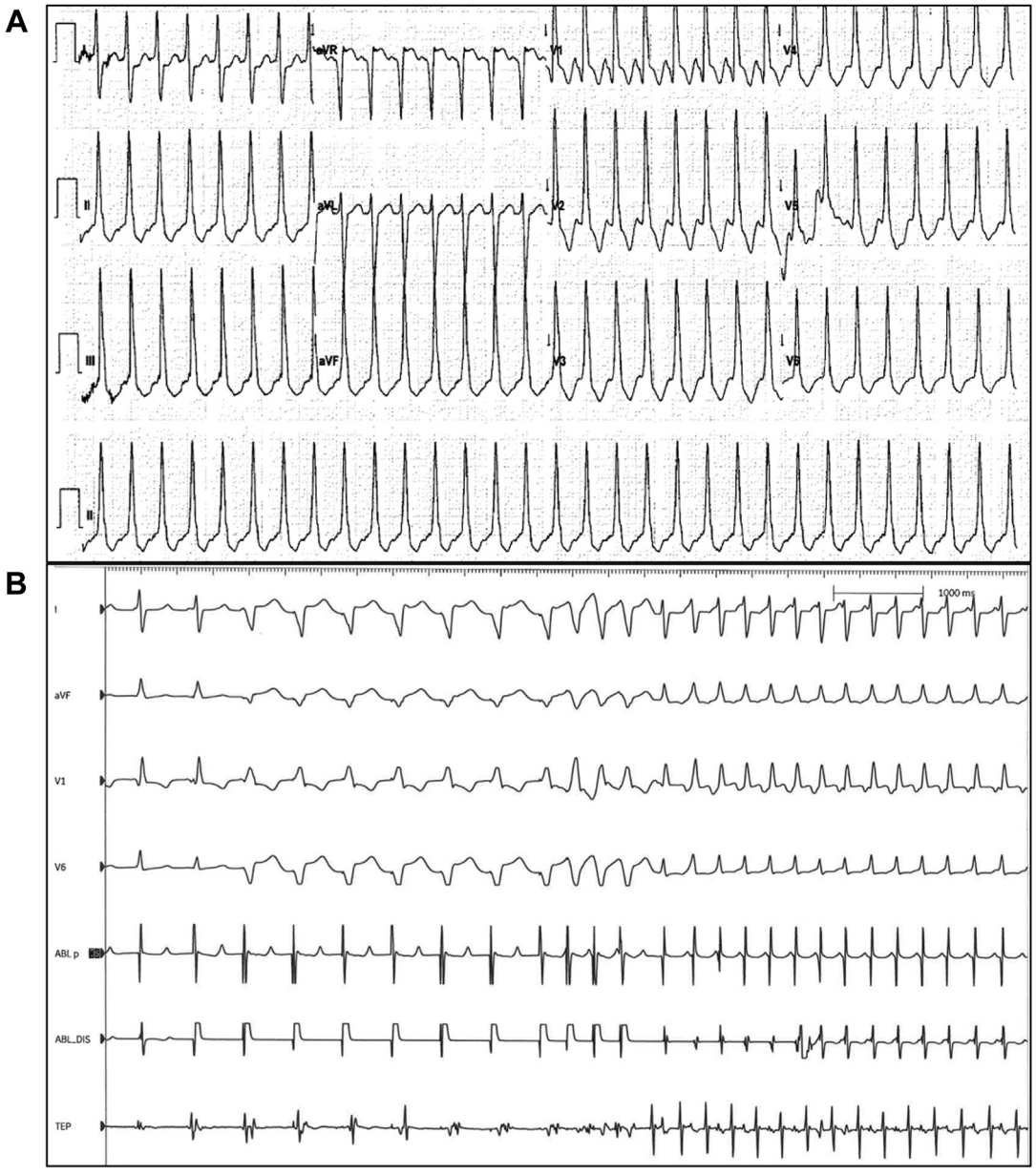

Figure 2 **A:** *Upper panel:* 12-lead electrocardiogram demonstrating long-RP tachycardia in a 27-year-old post-Fontan patient. Retrograde P waves are inverted in leads II and aVF, and upright in V1 and V2. **B:** *Lower panel:* Surface electrocardiogram tracings and intracardiac electrograms demonstrating induction of supraventricular tachycardia with ventricular extrastimulus testing. ABLdis = distal ablation; ABLp = proximal ablation; TEP = transesophageal catheter.

a 6-mm cryocatheter (Freezor Xtra 3, Medtronic, Minneapolis, MN), and multiple cryo-applications were placed (–75°C, 4 minutes) at the initial RF site; neither PR prolongation nor second-degree AV block were encountered. Post-ABL testing at baseline and on isoproterenol showed intact AV conduction and SVT remained noninducible. The only complication post procedure was abdominal discomfort from transhepatic access, resolving uneventfully. During follow-up of 1 year, the patient remained free of SVT off all anti-arrhythmic medications.

Discussion

AVNRT is one of the most common types of re-entrant SVT encountered in clinical practice. In patients with structurally normal hearts, ABL targeting the SP to the AVN within the TOK is associated with high rates of success and low rates of complications and tachycardia recurrence in most patients with either typical or atypical forms of AVNRT.[11] The electroanatomic approach to treat AVNRT involves ABL within the low–mid TOK, where local EGMs are consistent with stereotypical SP potentials,[2] and is currently employed by many electrophysiologists treating adult and some pediatric patients with structurally normal hearts. If initial applications are ineffective, additional ABL is usually performed nearby, sometimes extensively, and sometimes in a linear fashion from the tricuspid annulus back to the CS os.[2] A similar approach using cryoablation may be preferred when risk of complication (eg, AV block) seems higher, for example,

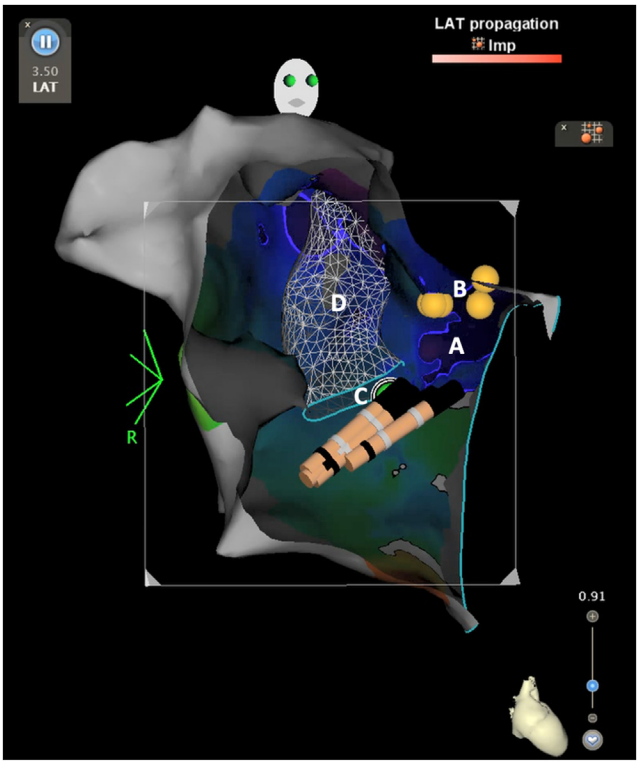

Figure 3 Clipped snapshot (CARTO 3, 3-dimensional system, Biosense Webster, Diamond Bar, CA; right anterior oblique view) of propagation mapping showing wavefront collision during low right atrial rhythm in a post-Fontan patient. **A:** Wavefront collision in the triangle of Koch (*thin light purple lines* surrounding *dark purple*, see Supplemental Video 1). **B:** His bundle electrograms (*yellow tags*). **C:** Sites of brief RF ablation (*covered green tag*) and catheter positions during cryoablation anterior to the coronary sinus (**D**, *mesh*).

when RF delivery causes rapid junctional acceleration,[12] as encountered in our case.

Adults with diverse forms of pre- and postoperative CHD may also undergo successful catheter ABL to treat AVNRT.[3,13] Reviewing a single center's experience with AVNRT ABL procedures in patients with CHD, Upadhyay and colleagues[13] described the univentricular subgroup as the most challenging. Relying on an electroanatomic approach, ABL was not attempted in 6 of 16 patients due to limited confidence of ABL targeting.[13] In a multicenter retrospective study, Papagiannis and colleagues[3] described outcomes of AVNRT ABL guided primarily by electroanatomic criteria in 103 patients with 2-ventricular anatomy and 6 with univentricular hearts. There were more frequent ABL failures in patients with complex 2-ventricular anatomy, univentricular hearts, or atypical AVNRT. In addition, complete AV block occurred in 4 patients with complex 2-ventricular anatomy and 1 with a univentricular heart.

Technological advances, including 3D EP mapping, widespread experience, and evolving techniques, have enhanced understanding of the pathophysiology of AVNRT, improved precision of SP mapping, clarified relevant anatomy and reduced fluoroscopic exposure to patients and laboratory personnel.[13,14] As a consequence, many practitioners treating children and patients with CHD and attendant distorted anatomy and atypical endocardial signals are moving away from the traditional electroanatomic approach for AVNRT.

One 3D technique evaluates for a low-voltage "bridge" within the TOK to indicate the potential location of the SP.[4] However, in treating pediatric patients with structurally normal hearts, Aartsen and coworkers,[7] using the Abbott mapping system, reported such bridges are either not routinely identifiable or appear in regions where ABL may not be advisable. This observation parallels our experience using the CARTO 3 system in similar patients, in adults with CHD status post 2-ventricular surgical repair[15] and the Fontan patient described herein.

More recently, dense endocardial EP mapping has facilitated visualization of wavefront activation and propagation patterns within the TOK and guided targeting of the SP.[7] In patients with structurally normal hearts, the successful site for ABL is often located in a zone slightly superior to where wavefronts collide within the TOK during sinus rhythm,[7] We have seen that a patch of abrupt conduction slowing is often demonstrated in this area, likely reflecting superior impulses conducting from local atrial myocardial fibers into the SP.[9,15] Targeting this patch for ABL has led to successful elimination of SP conduction and associated arrhythmias.[9,15] In the current case, wavefront propagation during the patient's prevailing low right atrial rhythm also showed a collision zone within the TOK. Importantly, the patch of abrupt conduction slowing was inferior not superior to collision, upside down from what is usually seen when impulses enter the TOK from above during sinus rhythm. In our patient, initial RF applications targeting this slow patch were limited due to rapid junctional acceleration that could portend AVN injury; perhaps targeting the zone just above wavefront collision, often effective after mapping propagation in the TOK during sinus rhythm, would have led to AV nodal injury. Fortunately, antegrade AV conduction remained at baseline, and yet these applications clearly blunted SVT inducibility. Cryoablation applications in this same area for "consolidation" were delivered uneventfully. The patient had no acute complications from ABL and she has not had SVT during 1 year of follow-up.

Conclusion

Patients with AVNRT and complex CHD present significant challenges for successful ABL therapy. We describe the first case of a patient with univentricular anatomy status post Kawashima Fontan palliation in whom the relevant anatomy and location of the SP to the AVN were mapped using wavefront propagation to identify the SP to the AVN, and AVNRT was eliminated with catheter ABL uneventfully via transhepatic and transbaffle access.

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Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.08.029.

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